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Uracil DNA Glycosylase Rabbit mAb

Catalog # AP78312

Product Information

Application	WB, IHC-P, IF, FC, ICC
Primary Accession	P13051
Reactivity	Rat, Human, Mouse
Host	Rabbit
Clonality	Monoclonal Antibody
Isotype	IgG
Conjugate	Unconjugated
Immunogen	A synthesized peptide derived from human UNG
Purification	Affinity Purified
Calculated MW	34645

Additional Information

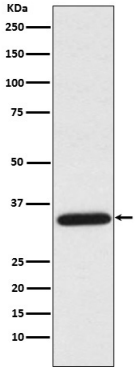
Gene ID	7374
Other Names	UNG
Dilution	WB~~1/500-1/1000 IHC-P~~N/A IF~~1:50~200 FC~~1:10~50 ICC~~N/A
Format	Liquid in 10mM PBS, pH 7.4, 150mM sodium chloride, 0.05% BSA, 0.02% sodium azide and 50% glycerol.
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.

Protein Information

Name	UNG {ECO:0000255|HAMAP-Rule:MF_03166}
Function	Uracil-DNA glycosylase that hydrolyzes the N-glycosidic bond between uracil and deoxyribose in single- and double-stranded DNA (ssDNA and dsDNA) to release a free uracil residue and form an abasic (apurinic/apyrimidinic; AP) site. Excises uracil residues arising as a result of misincorporation of dUMP residues by DNA polymerase during replication or due to spontaneous or enzymatic deamination of cytosine (PubMed:12958596, PubMed:15967827, PubMed:17101234, PubMed:22521144, PubMed:7671300, PubMed:8900285, PubMed:9016624, PubMed:9776759). Mediates error-free base excision repair (BER) of uracil at replication forks. According to the model, it is recruited by PCNA to S-phase replication forks to remove misincorporated uracil at U:A base mispairs in nascent DNA strands. Via trimeric RPA it is recruited to ssDNA stretches ahead of the polymerase to allow detection and excision of deaminated cytosines prior to replication. The resultant AP sites temporarily stall replication, allowing time to repair the lesion (PubMed:22521144).

	Mediates mutagenic uracil processing involved in antibody affinity maturation. Processes AICDA-induced U:G base mispairs at variable immunoglobulin (Ig) regions leading to the generation of transversion mutations (PubMed:12958596). Operates at switch sites of Ig constant regions where it mediates Ig isotype class switch recombination. Excises AICDA-induced uracil residues forming AP sites that are subsequently nicked by APEX1 endonuclease. The accumulation of staggered nicks in opposite strands results in double strand DNA breaks that are finally resolved via non-homologous end joining repair pathway (By similarity) (PubMed:12958596).
Cellular Location	[Isoform 1]: Mitochondrion

Images



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